mal <20) and lupus anticoagulant, with laboratory confirmation 6 weeks later. From 1999 she presented a chronic symetrical polyarthritis that was treated with steroids 10 to 20 mg/day. At the end of 2000, with 20 mg/day of prednisolone, laboratory findings were: erythrocyte sedimentation rate (ESR) 13 mm/1sthour; C-reactive protein < 3 mg/L, albuminemia 32 g/L (normal range 30.7 –50.4), immunoglobulin (Ig) G 3.5 g/L (normal range 6.75-12.8), IgA2.8 g/L (0.8-3.4), IgM 1.9 g/L (0.6-1.9). Pulmonary X-ray revealed a round regular opacity at the top of the right lung of 15 mm confirmed on CT-scan, without mediastinal adenopathy, suggesting a granulomatous lesion.

The diagnosis of CVI was retained and the patient received IVIG (40 g on one day every 4 weeks) resulting in a dramatic improvement of polyarthritis. Steroids were progressively tapered and stopped in December 2001. The size of the pulmonary granuloma decreased to 5 mm at the same time. In August 2002, she presented a Quincke oedema during the 14th infusion of IVIG. The search for anti-IgA antibodies was positive. Due to a relapse of sinusitis, we proposed IgA depleted IVIG (40 g on one day every 4 weeks). Before the second infusion, the level of total Ig was 6.5 g/dL (normal range: 7.0–15.0). Ten days after this second infusion, she complained of a relapse of polyarthritis. Laboratory findings were as follow: ESR 76 mm/1sthour; C-reactive protein 51 mg/L. Electrophoresis showed hypoalbuminemia (30 g/L) but Ig were in the normal range (10.2 g/dL). Despite a new perfusion of depleted IgA-IVIG, her clinical condition deteriorated, justifying starting prednisone again, 30 mg/day. Steroids were progressively tapered but the polyarthritis remained cortico-dependant despite regular IgA-depleted IVIG infusions.

This is the first report of an association of antiphospholipid syndrome and CVI. The relationship between systemic lupus erythematosus and CVI has been established for several years and it could be supposed that those beween APS and CVI are similar (6, 7). The present case suggests that IVIG could have a favorable impact on pulmonary granulomatous lesion. Two previous reports have also shown resolution of cutaneous granulomatous lesions and polymorphic light eruption with IVIG, suggesting, in addition to our case, that it has steroid-sparing and immunosuppressive effects in patients with CIV(4, 5).

Finally, we noticed a lower efficacy against rheumatologic manifestations of IgA-depleted IVIG than of non-depleted IVIG. As the only difference between these two types of immunoglobulin preparations lies in the level of IgA (2.1 mg/ml for non-depleted IVIG and 2.2 µg/ml for IgA-depleted IVIG) it is suggested that IgA played a role in the control of immune disease in our patient. *In vitro* studies have shown that IgA antibodies had a better antiinflammatory activity and regulates complement activity (8, 9), and that IgA/IgM enriched IVIG may be a more potent immunomodulator than pure IgG preparations (10). Our observation suggests that the IgA composition of immunoglobulin preparations could also lead to a difference in antiinflammatory effects in some patients.

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Development of myositis after hepatitis C virus infection

Sirs,

Persistent hepatitis C virus (HCV) infection leads to the development of chronic hepatitis, cirrhosis, hepatocellular carcinoma, and a wide spectrum of extrahepatic manifestations. Viruses have long been suspected as potential etiologic or triggering agents in the pathogenesis of many autoimmune diseases. Some immunological disorders have a well established association with HCV infection, the presence of cryoglobulins being the most common (1-3). An association between polymyositis/dermatomyositis (PM/DM) with lung fibrosis and chronic HCV infection has previously been reported (4-6). We recently observed a patient who developed myositis in association with chronic HCVinfection.

The patient, a 68-year-old woman, presented with a 4-week history of progressive muscle weakness of upper and lower limbs, and weight loss (10 kg). Seven days before she was unable to rise from her bed or to walk. She had a 4-year history of histologically confirmed liver cirrhosis and HCV seropositivity. Thirty years ago she had been operated on and received a blood transfusion. She was seen in our rheumatology unit in July 2004. On presentation the patient had a blood pressure of 200/100 mmHg and a normal temperature. No skin rashes or lymphadenopathy were found. Head and neck examination revealed non-icteric sclerae. There was truncal vascular spiders. The spleen was palpable. Ascites was absent. There was severe proximal muscle weakness with marked wasting. Laboratory test results showed hemoglobin 15 g/dl, leukocytes 11.300/mm^3 and 23,000/mm^3, alanine aminotransferase (ALT) 310 IU/L and 867 IU/L, aspartate aminotransferase (AST) 1,626 IU/Land 924 IU/L, lactate dehydrogenase 761 IU/L and 1.980 IU/L, and gammaglutamyl-transferase (GGT) normal (49 IU/L). Creatine kinase (CK) was increased at 40,000 IU/L. Total bilirrubin was 1.6 mg/dl; and albumin and alkaline phosphatase were normal. Serum creatinine was elevated (6.9 mg/dl) and there was hyperkalemia (7.6 mmol/L). Serum level of alpha-fetoprotein was within nor-

mal limit. HCV antibody was positive by a third generation ELISA. Muscle biopsy demonstrated cellular necrosis, regenerating fibers and inflammatory infiltrate. A lumbar puncture was normal. The patient was treated with antibiotics and corticosteroid. Because of her respiratory distress intubation and mechanical ventilation were required. A dialysis procedure was performed. She died on the fourth day of hospitalization.

Since its discovery in 1989, HCV is more than just a liver disease (7). Chronic HCV infection is associated with an array of autoimmune laboratory and clinical manifestations (2, 8). HCVmay represent a chronic stimulus for the immune system and/or direct invasion and replication of the virus in extrahepatic tissues (1).

The most useful enzymes in diagnosis and prognosis of PM/DM are CK and aldolase. The AST, ALT, and LDH enzymes may appear in increased amounts as well. These 3 enzymes share a site of origin in both muscle and liver. Our patient had HCV infection for years prior to the emergence of PM. She presented with clinical and histologic features of PM, no rash involvement, and raised CK levels which were related with acute renal failure. Further controlled studies are justified to determine an association between HCVinfection and PM, thus eventually leading to a better understanding of mutual relationship between virus and myositis.

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A new case of dermatomyositis following the rupture of a silicone gel breast implant

Sirs,

We read the interesting report of two cases of dermatomyositis which occurred in patients who had undergone silicone gel filled breast implant for cosmetic reason (1); in the first case the disease was diagnosed 6 months after breast augmentation and in the second one after 8 years; in the latter one chest HRCT demonstrated retraction of the prosthesis. HLA typing showed identity in one of two aplotypes of class II antigens: HLA-DRB1* [0301], HLA-DQB1* [0201]. The first patient strongly ameliorated after prosthesis removal, but required chronic immunosuppressive therapy to control the disease; the second patient refused the explantation of breast prosthesis.

Recently we have observed a case of dermatomyositis in a 57-year-old woman who first underwent silicone gel augmentation mammoplasty for cosmetic reason in 1970; in November 2003 the mammary prosthesis were substituted for silicone spreading following capsular rupture. Six months later she complained of painful proximal muscle weakness and periorbitary edema. CPK level was 8026 U/L; antinuclear antibodies were positive at dilution 1:1280 with speckled pattern; anti-ENA antibodies tested by ELISAwere negative. The extensive search of an occult tumour resulted fruitless. Muscle biopsy showed some atrophic cells, necrosis and inflammatory infiltrate. HLA typing showed HLA-A [24, 34], HLA-B [8(Bw6), 18(Bw6)], HLA-Cw [07, 12]; HLA-DRB1*[0301], HLA-DQB1* [0201]. The patient was treated with immunoglobulin (0.4 g/Kg/day for 5 consecutive days), prednisone at the initial dosage of 50 mg/day and azathioprine 100 mg/day with good result; the dosage of prednisone was then reduced to 25 mg/day.

During the last years the relationship between silicone prosthesis implant and induction of connective tissue diseases has been largely investigated with an initial enthusiasm; obviously the discovery of an etiologic factor or of a trigger involved could allow to increase our knowledge in this complex and intriguing context and could favour the comprehension of the pathogenetic mechanisms which lead to the damage in some rheumatic diseases whose etiopathogenesis remains still obscure. Nevertheless epidemiological studies have so far not shown an increased incidence of connective tissue diseases in women with silicone breast implants (2-7), even in case of prosthesis rupture (8); instead conflicting data have been collected about a possible relationship between silicone gel breast implant rupture and fibromyalgia (9, 10) as well as silicone breast implant and symptoms evocative of rheumatic disorders (11). Moreover, an increased frequency of low titre positive ANA, but not of high titre ANA in patients with silicone breast implants compared to controls has been observed (6).

On the grounds of the results of the abovementioned epidemiological studies silicone is still largely used in cosmetic surgery; nevertheless these reports do not allow to exclude the possibility that in susceptible individuals the stimulation of the immune system by silicone may induce the appearance of a connective tissue disease; silicone gel may escape from ruptured implants, became extracapsular thus representing an adjuvant for the development of an autoimmune disease. Our patient presented homozygosis for the same aplotypes of class II HLA antigens (DR and DQ) as the two patients previously described (1), supporting the hypothesis of a predisposing genetic background. In 1997 a report about two HLA-identical sisters bearing rheumatoid arthritis susceptibility genes that developed polyarthritis after silicone breast implants has been published; after removal of the mammary prosthesis one patient achieved a complete remission and the other one a significant improvement (12). The genetic background might condition the type of autoimmune disease that starts after silicone exposure.

Few other cases of dermatomyositis after insertion of silicone breast implants have been described (5, 13); among these we cite a case of dermatomyositis-like disease in which silicone particles within phlogistic areas have been observed in a specimen of the inflamed upper eyelid (14).

We have hypothesized that in our case the dermatomyositis was more probably provoked by the old ruptured breast implants than by the second insertion. At the moment however we are not able to propose its explantation.

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